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THE VETERINARY RECORD

The Journal of the British Veterinary Association

Patron: Her Majesty the Queen

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TWO OF THE 213 ILLUSTRATIONS

Fig. 72. Examination of the pulse at the ventral coccygeal artery in the ox.

Fig. 44. Severe swelling of the skin and subcutaneous tissues of the head and neck.

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Report of the Animal Health Services, 1955

THE annual report on the Animal Health Services in Great Britain* is in some respects one of the most important veterinary documents published in this country each year. Certainly it contains a wealth of information, and there can be few veterinary surgeons who will not find much to interest them within its covers.

Parts I to IV of the Report constitute the Report of Proceedings under the Diseases of Animals Act 1950. The report starts off with a statement on the transfer of certain animal health functions to the Secretary of State for Scotland. This transfer is consequent on the Government's decision to implement the recommendations of the Royal Commission on Scottish Affairs, in that the Secretary of State for Scotland should take over responsibility in Scotland for all animal health matters. However, to meet the obvious necessity for prompt action in outbreaks of epidemic diseases in animals the single control of operational measures against such diseases remains vested in the Ministry of Agriculture. An Order in Council to effect this dichotomy came into effect in 1955, and the report states that the requisite machinery is functioning smoothly, and the transfer itself was accomplished without difficulty. This is yet another example of the British genius for operating the involved compromise, and one hopes the new arrangements will be a source of gratification to Scottish *amour-propre*.

As far as foot-and-mouth disease was concerned, 1955 was outstanding in that only nine outbreaks occurred. This is the lowest incidence since 1918. Seven of these outbreaks were regarded as primary, in that they had no known connexion with any other outbreak in Great Britain. Six of these primary outbreaks were thought to have originated from imported meat and meat wrappers. The viruses concerned were types C and O. Certain administrative changes affecting control measures, following recommendations of the Departmental Committee on Foot-and-Mouth Disease, 1952–4, were put into effect. The standard size of an infected area was reduced from a radius of 15 to 10 miles, and the salvage of carcases for human consumption of apparently healthy in-contact animals was stopped. All veterinary surgeons practising within an infected area were also notified immediately of all outbreaks which occurred within the area.

During 1955 there were eight outbreaks of atrophic rhinitis. In all except one the pigs involved were pure-bred Landrace, the exception was a herd of cross-bred Landrace and pigs in contact with them. Seven of the outbreaks were in pigs or their progeny imported from Sweden or the Isle of Man. Of the 960 pigs slaughtered 106 (11 per cent.) were found to have lesions of the disease on *post-mortem* examination.

There was a definite increase in the number of outbreaks of anthrax, there were 764 outbreaks which is more than twice the number in 1954. The average mortality was 1·03 per outbreak in cattle and 1·12 in pigs. These two species provided most of the cases, although 29 mink died from the disease. Feeding-stuffs and fertilisers again appeared to be the most probable sources of infection. It was disturbing that over 15 per cent. of cases occurred where the carcases had been opened.

The fowl pest position cannot be said to have altered significantly. There were 906 outbreaks, one of which, in a West Sussex hatchery, resulted in infection of 108 premises, and the slaughter of 50,000 birds. The areas affected during the year were South Wales and South-west England, Norfolk, Lancashire and Essex. The laconic statement of these facts in the report gives no indication of the enormous amount of work undertaken by the veterinary staff of the Ministry in tracing and dealing with this disease ; but for their efforts the position would be much more serious and the cost to the industry would be very much greater than it is at the present.

There follows a fascinating account of 20 years' progress in the eradication of bovine tuberculosis. There is so much information in the 14 pages covering this subject that it is not easily summarised, and veterinary surgeons actively engaged in the eradication scheme will be well advised to read for themselves how their efforts are affecting the national position. By 1955 the position was very encouraging, over 6,000,000 (62 per cent. of all cattle) cattle were attested, and some 2,000,000 of these were in attested areas. Outside these

* Report on the Animal Health Services in Great Britain, 1955. Ministry of Agriculture, Fisheries and Food. Department of Agriculture, Scotland. London, H.M.S.O. 5s.

A

areas there remain 7,750,000 cattle, but more than half of these (about 4,000,000) were in attested herds. The number of cattle slaughtered in 1955 under the Tuberculosis Order of 1938 was 1,250, which, compared with 20,000 before the war, is another illustration of the steady progress made in eradication. In 1955 75 per cent. of the milk sold in this country was produced from herds regularly tuberculin tested.

The report goes on to state " there is every reason to hope that by the early 1960s bovine tuberculosis in Great Britain will, for all practical purposes, be a thing of the past," a justified, but unusually sanguine statement for a Government department.

To many readers, Part V will be the most stimulating part of the report for it contains accounts of the laboratory, research and investigation services. Here will be found interim reports of research and investigation into nearly all important problems likely to be met with in farm practice, and even the small-animal practitioner will find something of interest, for work goes on at Weybridge on the simultaneous vaccination of dogs against distemper and Rubarth's disease.

This part of the report is impossible to abstract and one can only list some of the subjects discussed. These include, among others, brucellosis, vibriosis, Johne's disease, salmonellosis, nematodiriasis, parasitic bronchitis, coccidiosis, fluorosis, metabolic disorders, and bowel oedema in pigs. There is a hint of an association between certain haemolytic strains of *Esch. coli* and bowel oedema in pigs.

Among the more exotic items of information are a mention of high mortality in young eider ducks, in Aberdeenshire, due to the filaroid worm *Polymorphus boschadis*, whose intermediate host apparently is the shrimp, *Gammarus pulex*, and some further additions to the plethora of Salmonella infecting poultry, *Salm. fresno* in ducks and *Salm. stanleyville* and *Salm. mbandaka* in turkey poults.

Within the pages of this prosaic report is a record of work done in which the veterinary staff of the Animal Health Division in particular, and the veterinary profession in general, may have justifiable pride.

In Brief

BOVINE CYSTICERCOSIS

Cysticercus bovis, the larval stage of the human tapeworm *Taenia saginata*, is fairly common in the tropics, and is being more frequently reported in temperate countries such as Britain (Griffiths, 1950), Denmark (Jepsen & Roth, 1952), Belgium (Gregoire *et al.*, 1956), and the U.S.A. (McIntosh, 1956). The incidence in British cattle according to the Ministry of food varied between 0.21 and 0.58 per cent. during the period 1949-53. Of 500 abattoirs in operation under the Ministry only 200 reported the presence of *C. bovis*, and of these, 45 consistently found 0.81 to 3.47 per cent. of the carcases infected (Silverman, 1955). These cattle came from both home-bred and Irish store stock. Over 4,500 local slaughter-houses were reopened following decontrol in 1954 and records are more difficult to obtain, but the evidence suggests that the rate of infection has not lessened.

The chances of a meat inspector discovering *C. bovis* cysts in a carcase depends upon the number of inspection incisions and the thoroughness with which they are made and examined. The results of experimental infections suggest that (1) the masseters and head are not necessarily primary sites of election for the development of cysts; and (2) the state of degeneration of cysts in one part of the carcase is not necessarily indicative of the state of cysts in the same or other sites (Penfold, 1937; Jepsen & Roth, 1952; Silverman, 1956). Over 40 per cent. of the cases diagnosed at the Letchworth abattoir during the past six years were discovered solely from careful heart incisions, no cysts having been revealed in these cases, by masseter and tongue incisions (Jump, 1949-55).

It is suggested that cattle in this country become infected from drinking sewage-polluted water or from grazing pastures contaminated with droppings of sewage-infected sea-birds. Tapeworm eggs are resistant to normal sewage treatment processes and may pass out in sewage effluents to contaminate streams and rivers into which they are discharged (Penfold *et al.*, 1936; Vasilkova, 1944; Newton *et al.*, 1949; Jepsen & Roth, 1952; Wang & Dunlop, 1954; Silverman & Griffiths, 1955). It is known that seagulls feed at sewage plants and at seaside sewage outfalls and become contaminated with sewage products both by ingestion and physical contact. Tapeworm eggs are capable of passing through the gut of seagulls. (Götzsche, 1951; Silverman & Griffiths, 1955).

The incidence of infection in man appears to be on the increase although national statistics are difficult to get as the condition is not notifiable (Silverman, 1956). Closer co-operation between veterinarians, public health inspectors, and medical officers of health is greatly to be desired to improve meat inspection techniques, sewage treatment methods, and to publicise the desirability of thoroughly cooking all meat and meat products. It would appear that the veterinary surgeon should take an active lead to secure this end.

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Meningoencephalitis eosinophilica of Swine. I.—The Naturally Occurring Disease

BY

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Introduction

THIS condition, which was first described in Austria by Michalka in 1941, under the name of *Leptomeningitis eosinophilica*, has also been seen in Germany (Godgluck, 1952), Sweden (Hjärre, 1952, Isaakson, 1952), Denmark (Larsen, Moller & Thordal-Christensen, 1955), and Japan (Yamagiwa, 1955).

In Britain, it was first recognised in 1951 when an outbreak near Newmarket was investigated at the end of August of that year. Since that time it has been encountered with increasing frequency, possibly due to a greater volume of neuro-pathological material being examined.

The present paper contains an account of the original investigation and of a more recent outbreak. These, together, cover most of the clinical features of the disease as seen in the field, while the outbreak of February, 1955, is of further interest in that it was possible for the first time to establish an aetiological basis for the disease. The experimental reproduction of the syndrome and the detailed pathology will be dealt with in further publications.

Protocols

Outbreak A (August, 1951. D.B.)

A sow with a litter of nine piglets were the only pigs kept on the farm. A few days after the pigs were weaned, at eight weeks of age, one died suddenly. Two or three days later, seven of the remaining piglets were affected with "convulsions." One piglet died within 24 hours and the remainder of the affected animals within the week, with the exception of one which recovered. Though running with the rest throughout, one piglet showed no clinical abnormality whatsoever and continued to feed and behave quite normally.

Affected piglets would suddenly stand still with a strained, tense expression, followed in a few seconds by champing of the jaws, frothing at the mouth, violent muscular tremors producing shaking of the whole body, forced backward movements, and collapse. These fits recurred at frequent intervals, sometimes after only a few minutes, and during the intervals the piglets were apparently blind, wandering aimlessly round the pen.

One live piglet was removed to the laboratory for autopsy but no significant macroscopic changes were found. Cultures on sheep-blood agar from heart blood, liver, spleen, and lung were all negative as was a biological test carried out on mice using whole blood.

The brain was removed and fixed in 10 per cent. formol-saline for subsequent histological examination. This revealed a non-suppurative encephalitis characterised by perivascular cuffing and, to a lesser extent, neuronal degeneration. These changes were confined to the fore-brain and were most severe and extensive anteriorly. The vascular cuffs consisted chiefly (about 90 per cent.) of eosinophile leucocytes with a smaller number of round cells. No cellular inclusions or bacteria could be seen, nor was there any indication of the nature of the aetiological agent.

The history, clinical signs, autopsy findings, and the distribution, but not the type, of lesions in the brain suggested Aujeszky's disease, though it appeared unlikely that such a high mortality could be expected in weaned pigs with this disease. However, eight weeks later an opportunity occurred to obtain serum samples from the two survivors of the litter, one of which had never been clinically affected. In neutralisation tests carried out on the chorioallantoic membrane of embryonated eggs these sera completely failed to neutralise an egg-adapted strain of Aujeszky's virus. These sera also gave a negative complement fixation test for lymphocytic choriomeningitis.

Outbreak B (February, 1955. N.H.B.)

Two neighbouring farms received part of a consignment of a proprietary pig meal which, on subsequent analysis, was found to contain 7.2 per cent. of common salt.

On Farm 1 the food was fed to about 30 pigs of all ages, though it seems probable that for one day at least the meal was mixed with the last of the old consignment.

The method of preparing the food for the pigs on this farm was as follows: a two-gallon bucket was half filled with meal, and water mixed into the meal until the bucket was nearly full. Each store pig was allowed about 2 lb. of meal a day; no other water supply was available.

Nothing abnormal was noted in the behaviour of the pigs on the first or second day the meal was fed. On the third day two groups of stores aged 14 to 18

weeks showed signs of thirst in the morning. Little attention was paid to this as the pens were of the cottager type, small and warm inside, and the owner thought the pigs might have been too hot. At the evening feed two pigs were noticed to be staggering about and weak on their hind legs. The following morning another pig appeared blind. Later that morning two more pigs were observed lying on their bellies with legs outstretched and kicking frequently. One of the first pigs to become ill died that evening. By this time all the stores were refusing to eat the food; all appeared constipated and would drink urine or any other available fluid avidly. During the fourth night after the introduction of the salty food two more pigs developed fits and by the fifth morning three had died. The owner's veterinary surgeon, suspecting the food to be responsible, had all the pigs on the premises starved and instituted symptomatic treatment. No further losses occurred. Throughout the outbreak none of the breeding or fattening pigs showed any clinical abnormality.

On Farm 2 the food was given to 14 store pigs aged 15 weeks. These were housed in three lots, one lot comprising four pigs which were very well-grown for their age. The meal, at the rate of 2 lb. per pig per day, was mixed with boiled potato peelings and scraps to the consistency of thick porridge. No other water was supplied to the pigs. As on Farm 1, the trough space was inadequate for the number of pigs being fed.

The new consignment of food was first given at the evening meal and was fed for both meals on the following day. On the second morning one pig attempted to drink the fluid in the mixture but refused the solid portion of the food. Later in the day three pigs were observed to be staggering and "throwing fits." During the next 36 hours seven pigs died including all the group of four well-grown animals. At this stage the food was withdrawn, the pigs were treated and no further losses occurred.

Pigs dying on both farms were submitted for *post-mortem* examination but no significant macroscopic changes were seen and no pathogens recovered on bacteriological examination. Histopathological examination of the brain revealed the now familiar picture of *Meningoencephalitis eosinophilica*. In both the brains examined lesions were confined to the cerebrum and consisted of acute necrosis of the cerebral cortex with hydropic degeneration of the neurones and a vasculitis characterised by endothelial proliferation and spectacular perivascular infiltration by eosinophile leucocytes. In one, the necrotic changes predominated and in the other the proliferative changes were more pronounced, but in both it was remarked that lesions were more severe anteriorly and over the dorso-lateral parts of the cerebrum.

Discussion

Pathology

Since the definitive characters of this disease are purely pathological, it seems appropriate to discuss this aspect first, though it is not our intention to describe the histopathology of the condition in detail in the present paper.

The essential lesion in all the cases so far seen has been an acute necrosis of the grey matter of the brain, always localised and usually confined to those parts of the brain anterior to the corpora quadrigemina. This is usually associated with a thickening and proliferation of blood vessels in and around the walls of which are to be found considerable numbers of eosinophiles. In most cases it is the cerebral cortex which is attacked and usually the damage is most severe in the tissues bounding the depths of the sulci, the eosinophile infiltration commonly being conspicuous in the sulci also. Though the pathognomonic eosinophile infiltration is not present in every case and appears to depend on the age of the lesion at the time of death, it seems worth while to retain for the present the name *Meningoencephalitis eosinophilica* suggested by the Scandinavian workers since this refers to the characters which differentiate it from all other known conditions of pigs, and is to be preferred to the less apposite *Leptomeningitis eosinophilica* of Michalka and *Encephalitis eosinophilica suis* of Godgluck.

In the fixed brain it is possible, in those cases in which the cerebrum is severely affected, to see with the naked eye the zone of necrosis in the cerebral cortex midway between the white matter of the corpus medullare and pia mater, the zone being more pronounced round the depths of the sulci, but there is usually an absence of significant macroscopically observable changes elsewhere in the carcase.

There seems little doubt that *Meningoencephalitis eosinophilica* of swine as seen by us is identical with that first noted by Michalka and described in more detail by Larsen *et al.*, and Yamagiwa. Through the courtesy of Professor Hjärre, one of us (J. T. D.) has been able to compare histological material from British and Swedish cases and to confirm that, in so far as the histological picture is concerned, the condition encountered in the two countries is the same.

Clinical Signs

Clinically the condition is characterised by nervous signs principally of cerebral origin, compulsive movements, walking in circles, pressing the head or nose against walls, muscular tremors, champing of the jaws, apparent blindness, and epileptiform convulsions.

Though signs attributable to damage to the central nervous system are common in the infectious encephalitides of the pig encountered in this country, such as swine fever, Aujeszky's disease, and streptococcal choriomeningitis, the spinal and cerebellar components are usually more marked than in *Meningoencephalitis eosinophilica*, and, except in Aujeszky's disease, the incidence of epileptiform convulsions is normally low. Aujeszky's disease, however, will not usually produce clinical disease in pigs over weaning age. Oedema disease may present similar nervous signs, but here again the low incidence of epileptiform convulsions and the early onset of paresis are of assistance in differential diagnosis.

Similar clinical signs have been described by many authors in outbreaks of salt poisoning in pigs (see review by Worden, 1941), and also in experimental

poisoning by sodium chloride (Bohosiewicz, 1953; Bohstedt & Grummer, 1954).

Many of these reports are in close agreement on the nervous manifestations of salt poisoning, and the composite picture they present bears a striking resemblance to *Meningoencephalitis eosinophilica*.

Of the non-nervous signs, thirst is the most prominent clinical feature but this may well pass unnoticed if fluid is present in the pigs' trough. Diarrhoea is not usually present and, indeed, constipation is the more common finding.

Aetiology

Michalka (1941) did not advance any theory as to the cause of the condition he described, but Fankhauser & Wyler (1953), reviewing the literature, suggested that it might be an allergic manifestation as do also Larsen *et al.* (1955), though both the latter group of workers and Hjärre (1953) noted the association of large amounts of salt in the diet with the naturally occurring disease. Quoting Hjärre's cases, McNutt (1953) suggests Vitamin E deficiency as a possible cause, while Yamagiwa (1953) considered that " defining it as a sort of infectious encephalitic disease can be permitted at present."

In this country the occurrence of the condition in a considerable number of pigs on the same premises at first suggested an acute transmissible disease. However, the pathological changes in the central nervous system did not resemble those described in any known infectious disease, bacteriological examination was consistently negative and the sera of recovered cases did not contain antibodies to Aujeszky's virus or the virus of L.C.M. Intracerebral inoculation of brain material from another outbreak into two pigs at Weybridge produced no clinical or pathological abnormality (Hudson & Done, 1951).

Experiments in this laboratory had shown (Done & Joyner, 1953) that extracts of *Trichomonas suis* when injected intravenously will produce in the pig a severe shock reaction with cyanosis, respiratory failure, and cardiac disturbance, but when, in an attempt to simulate the pathological changes found in the naturally occurring disease by the production of an anaphylactoid reaction in the brain, this material was inoculated intracerebrally, no clinical or histological reaction resembling *Meningoencephalitis eosinophilica* could be detected.

However, as a result of the investigations of Outbreak B described above, interest in common salt as a possible aetiological agent was renewed and it was appreciated for the first time that the toxicity of the salt might be influenced by the amount of water available, and that the reluctance of pigs to eat salty food might be at least partially overcome by competition between pigs due to the provision of inadequate trough space. The significance of the salty meal as the precipitating cause of the disease in this instance was emphasised by the recovery of the remaining pigs when the diet was changed.

In a personal communication to one of us (J. T. D.) Innes (1952) suggested that this type of lesion might be caused by sodium chloride poisoning, but this possibility was rather discounted at the time, in view of the known difficulty in poisoning pigs with common salt (Wautie, 1939; Slavin & Worden, 1941) and the fact that pigs involved in naturally occurring outbreaks of *Meningoencephalitis eosinophilica* were usually fed on proprietary meals of controlled composition.

By simulating the method of feeding obtained on Farm 1 of Outbreak B, Done, Harding & Lloyd (to be published) have succeeded in reproducing the clinical and histological features of *Meningoencephalitis eosinophilica* with diets containing between 5 and 9 per cent. of common salt on a dry-matter basis.

In Poland, Bohosiewicz (1953), and in America, Bohstedt & Grummer (1954), have been able to poison pigs with sodium chloride by simultaneously restricting water intake. In both cases nervous signs were produced of the type described in naturally occurring salt poisoning and *Meningoencephalitis eosinophilica*, but Bohosiewicz did not examine histologically the brains of his pigs, and although Bohstedt & Grummer report that the brains of their pigs were " apparently normal," it seems unlikely that a detailed pathological examination was carried out.

It may be that disturbance of water balance rather than specific toxicity of sodium chloride is the critical factor in the genesis of *Meningoencephalitis eosinophilica*, and that the way in which this is produced is relatively unimportant, but there is no doubt that a high sodium chloride intake, when combined with restriction of water intake, can produce the disease, and it seems probable that this is the most common cause of the condition as encountered in the field in this country.

Summary

(1) Two unrelated outbreaks of *Meningoencephalitis eosinophilica* in pigs are described and the clinical and pathological features of the disease are summarised.

(2) The possible aetiology of this condition is discussed, with particular reference to salt poisoning.

(3) It is considered that though other agents may possibly be able to set up the disease, the most common cause in this country is probably an excess of common salt in the diet combined with restricted water intake.

Acknowledgments.—To Messrs. D. L. Grant, B.SC., M.R.C.V.S., and F. G. Greer, M.R.C.V.S., who brought to our attention outbreaks A and B respectively. To Mr. M. Gitter, M.R.C.V.S., for bringing to our notice and translating the paper by Bohosiewicz. To Mr. W. Mansi, M.V.SC. (CAIRO), DIP. BACT., for carrying out the complement-fixation tests against L.C.M.

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(*Concluded at foot of col.* 1 *overleaf*)

Legal Pitfalls of Veterinary Practice

BY

CHARLES STEVENS

V.—CONTRACT AND WARRANTY

PREVIOUS articles have dealt with the liability of the veterinary surgeon in tort but pitfalls exist also in the field of contract. Without going into legal technicalities it can be said generally that a contract consists of a promise for which there is a consideration, the promise being of two parts, offer and acceptance. When a veterinary surgeon undertakes the treatment of a client's animals and his services are accepted by the client, or conversely when the client asks him to undertake the treatment and the veterinary surgeon accepts this duty, a binding promise is made, enforceable at law, the consideration being the fee the veterinary surgeon expects to receive.

This seems quite straightforward and unlikely to give rise to difficulties, but any conditions or warranties that become attached to the promise are by law incorporated into the contract and may be enforced to the detriment of the veterinary surgeon. It was stated in the first article that the veterinary surgeon should avoid offering to cure an animal of disease because this would be accepted as a warranty or guarantee, and if a cure were not effected an action for breach of contract may well lie against him. Offers, conditions or warranties regarding treatment may sometimes be seized upon by the client.

In one case when castrating a bullock the veterinary surgeon said he was trying out a new method and jokingly undertook to stand the cost of the animal if it died. Unfortunately the animal did die and subsequent investigation showed that the method of castration used was one widely recognised by the profession and that the veterinary surgeon had not been guilty of negligence. Normally the death of the animal would not have given rise to a claim but the undertaking given by the veterinary surgeon to stand the cost if the animal died was held to be part of the contract, and he had to pay a considerable sum of money to the owner.

It should be noted that, contrary to general belief, except for a few statutory cases with which the veterinary surgeon is not often concerned, a contract is binding whether it be written or verbal, and therefore any statements made to a client, especially if made in the presence of witnesses, can be attached to the contract and may be used against the veterinary surgeon at a later date. The advice previously given to be discreet in speech as well as careful in action cannot be too often emphasised.

On many occasions a veterinary surgeon will be asked to given an opinion about an animal, and here it is essential not to be too dogmatic, especially if the question of purchase and sale is involved as his opinion acted upon by one of the other parties may well be incorporated as a condition or warranty in the contract between them. If there is any doubt whatsoever a firm opinion should be avoided and the veterinary surgeon should state plainly that " I think according to the best of my knowledge . . ." Another point about giving an opinion about an animal is to make quite sure that if any statement is given in writing the words " at the time of my examination of this animal " are added before the opinion is set out.

In one example that came before the Mutual Defence Society a veterinary surgeon had been asked for an opinion on an eight-year-old gelding before it was sold, and he used the words " The animal is sound." without any qualification whatsoever. The animal was sold and shortly afterwards was found unsound by a second veterinary surgeon. The purchaser threatened the vendor with an action and he immediately brought in the first veterinary surgeon under warranty and breach of contract. An expert was provided by the Society who confirmed unsoundness at the time of his examination. The only course was for the Society to settle out of court.

To conclude, a word of warning should be given to the veterinary surgeon concerning contracts of insurance. These are regarded at law as contracts made in perfect good faith and any non-disclosure or evasion of a clear statement of fact may well void a policy with possible heavy loss to the veterinary surgeon.

SALE OF RAW MILK

The Medical Officer of Health told the Tenbury Wells, Worcestershire, Rural Council that in recent months he had received several reports of children being taken ill with tuberculosis of bovine origin because their parents bought raw milk from farmers. Tenbury was a designated area, and he gave a warning that any farmer who sold milk other than a specially designated type, even to his next-door neighbour, rendered himself liable to prosecution.

Meningoencephalitis eosinophilica **in Swine**—*concluded.*

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Hypomagnesaemic Tetany in Calves Fed on Milk Diets

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DURING the last few years, the number of reported cases of hypomagnesaemic tetany in calves has increased considerably. This may be due either to an increase in incidence, or, more probably, to a more widespread recognition of the disease. Many of these cases have been confirmed by serum analysis, and the results indicate that the condition occurs in animals from about eight weeks to several months of age. It appears most frequently in calves between two and four months of age receiving mainly whole milk rations. Cases have been reported, however, where the ration has consisted of milk substitutes ; of milk, concentrates, and hay ; and in suckling calves running at pasture with their dams.

The early work of McCandlish (1923) and Huffman & Robinson (1926) indicated that calves cannot be reared to maturity on whole milk alone due to the onset of tetany during growth. The latter authors found that onset of tetany was often associated with hypocalcaemia. Serum magnesium levels were not estimated in their early experiments. Later, Duncan, Huffman & Robinson (1935) and Huffman & Duncan (1956) demonstrated that when calves were maintained on a whole milk ration their serum magnesium levels gradually fell, and that tetany was always associated with low magnesium levels, while serum calcium levels were normal in many cases. Subsequently, Duncan, Lightfoot & Huffman (1940) also showed a marked seasonal drop in serum magnesium values of calves during the transition from winter to summer conditions and they attributed this to climatic effects.

Recently, Blaxter, Rook & MacDonald (1954a) have described the syndrome produced in calves fed on artificially prepared milk with a low magnesium content, and Blaxter & Sharman (1955) have reported investigations on some field outbreaks of hypomagnesaemic tetany in calves.

The present report describes the results of the experimental production of tetany in calves fed on whole milk diets. The results obtained are compared with those for calves maintained on a supply of artificial milk having a low magnesium content. A preliminary communication on part of the work has already been published (Parr & Allcroft, 1953a).

Experimental

Procedure

The work to be described involved a total of 35 calves and was carried out in two separate experiments. In the first of these, 20 calves were divided into five groups each consisting of four animals. In the second, 15 calves were allocated to four groups, one of which contained six animals, with three in each of the remaining groups. Ayrshire and Friesian calves of both sexes were employed. They were kept in loose-boxes and bedded on peat to prevent ingestion of roughage other than that fed experimentally. The rations fed to each group are summarised in Table I. These rations were introduced when the calves were a week old, but the flaked maize and hay fed to Groups 3 and 4 respectively were not completely consumed until the calves were about a month old.

The vitamin B mixture added to the milk of Groups 5, B and D was that suggested by Kastelic, Bentley & Phillips (1950). All calves which received whole milk were dosed with a synthetic preparation of vitamins A and D*, supplying 15,000 I.U. of vitamin A and 3,000 I.U. of vitamin D per week. In the first experiment they also received 50 mg. of vitamin E per week. Iron sulphate and copper sulphate were also added to the whole milk to prevent the development of anaemia which otherwise occurred.

The calves were bled regularly at weekly intervals for the estimation of serum calcium and magnesium levels. They were also bled at intervals for the estimation of blood pyruvic acid. Additional samples for calcium,

*Radiostoleum.

TABLE I
DAILY CALF RATIONS

Group	Whole milk, per cent. bodyweight	Artificial milk, per cent. bodyweight	Flaked maize, lb.	Hay low in magnesium, lb.	Vitamin B supplement	Antibiotics mg.	Mg intake, mg. per kg. bodyweight
Experiment 1							
1	10	—	—	—	—	—	10
2	20	—	—	—	—	—	20
3	10	—	1	—	—	—	12
4	10	—	—	1	—	—	14
5	—	10	—	—	+	—	5–10
Experiment 2							
A	10	—	—	—	—	—	10
B	10	—	—	—	+	—	10
C	10	—	—	—	—	100	10
D	—	10	—	—	+	—	5

magnesium and pyruvic acid estimations were taken as required when clinical symptoms became apparent.

At the end of the experimental period the surviving calves were killed. The magnesium contents of tissues from these and from the animals which had died of tetany in the course of the experiments were estimated. In the first experiment four muscles, liver, kidney and the tibia from each calf were examined. Corresponding samples were also obtained from five calves which had been weaned on to a ration which consisted entirely of hay and cake. These calves had normal serum magnesium and calcium levels when they were killed. In the second experiment analysis of the soft tissues was omitted but samples of the radius, ribs and thoracic vertebrae were obtained in addition to the tibia.

Materials

In the first experiment, the artificial milk was prepared according to the formula of Wiese, Johnson, Mitchell & Nevens (1947) except that the lard was replaced by margarine, and of course, the magnesium content was reduced to levels varying between 5 and 10 mg. per 100 ml. of the milk. In the second experiment it was prepared according to the formula developed by Kastelic *et al.* (1950) except that again the magnesium content was reduced to give a final concentration of 5 mg. per 100 ml. in the milk. This was prepared daily in the laboratory as required.

The whole milk used was bulked milk from many cows. Samples taken at weekly intervals gave values varying between 9 and 14 mg. of magnesium per 100 ml. with an average value of 10 mg. per 100 ml.

The low magnesium hay fed to Group 4 was prepared by soaking hay in 1 per cent. hydrochloric acid for 24 hours. At the end of this period it was washed until free from the acid, drained, and then dried in a hot-air oven. It was not found possible to free the hay entirely of magnesium and generally the magnesium content was reduced from an initial level of about 0·15 per cent. to a final content of 0·05 per cent. The flaked maize used was found to contain an average of 0·03 per cent. of magnesium.

Analytical Methods

Serum calcium was estimated by the method of Clark & Collip (1925) and serum magnesium by the method of Denis (1922) employing the colorimetric finish of Fiske & Subbarow (1925) for phosphorus. Calcium in bone was determined by the A.O.A.C. standard method. The filtrate and washings from the calcium precipitation were made up to 250 ml. and 5 ml. aliquots taken for the estimation of magnesium by the method employed for serum.

Pyruvic acid in blood was determined by the method of Friedemann & Haugen (1943).

Results

In general the rations were taken without difficulty and, clinically, reasonable health and growth were maintained until the animals were about eight weeks old. The few exceptions were calves which scoured, particularly when milk intakes were being increased to bring them into line with the stipulated amounts. A lack of roughage was no doubt a contributory factor. Six of the 35 calves used were lost from the experiments through this cause. In some of them scouring produced fatal results, while in others it resulted in cessation of growth and the animals were removed. *Post-mortem* examination of the fatal cases usually revealed decomposing milk in the rumen. Bacterial examination for pathogenic organisms gave negative results.

The animals in Group 2 did not drink all their allotted amounts of milk from about 16 weeks of age onwards, due to the large volume involved.

Serum Magnesium Levels

The initial serum magnesium levels were 2·0 to 2·5 mg. per cent. These began to fall shortly after the commencement of the experimental rations, and by the third and fourth weeks many calves showed serum levels below 1·8 mg. per cent., which is the lower end of the normal range. This fall continued until values as low as 0·4 mg. per cent. were reached. Examples are illustrated in Figs. 1 and 2. In the first experiment symptoms of hypomagnesaemic tetany were first observed at about eight weeks of age in one of the calves in Group 1, which was fed on whole milk only at the rate of 10 per cent. of its bodyweight per day. This was followed by similar symptoms in some of the calves in Group 3, which received whole milk with

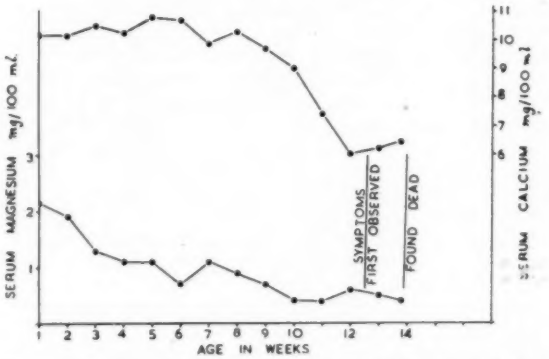

FIG. 1.—Serum calcium and magnesium levels of a calf in Group 3, fed on milk, 10 per cent. of its bodyweight per day, and flaked maize.

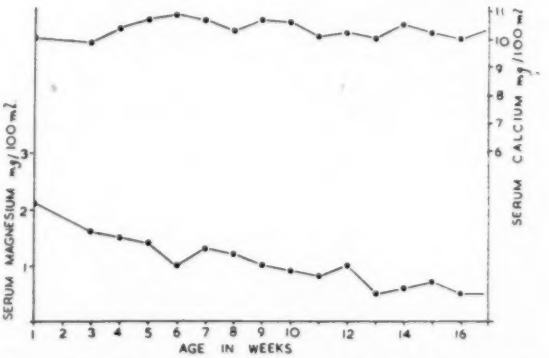

FIG 2.—Serum calcium and magnesium levels of a calf in Group 4, fed on milk, 10 per cent. of its bodyweight per day, with acid-extracted hay. This calf showed no clinical symptoms and was eventually slaughtered.

flaked maize, and in some in Group 5 which were fed on artificial milk. Twenty-one weeks elapsed before symptoms were first observed in a calf in Group 2, which received whole milk only at 20 per cent. of their bodyweight per day. There was a considerable difference in the ages at which symptoms appeared in individual animals in the same group and some did not show symptoms. No clinical symptoms were seen in any of the calves in Group 4, which were fed on whole milk and hay having a low magnesium content. These animals were eventually slaughtered. The ages at which death occurred, either from tetany, or by slaughter, are given in Tables II and III.

Serum Calcium Levels

In many cases the onset of tetany could be predicted from a rapid drop in the serum calcium values, as shown in Fig. 1. Serum calcium levels of cattle usually lie within the range 8·5 to 11·5 mg. per cent., with an average value of about 10 mg. per cent. In young calves, however, the range appears to be higher, from 10·0 to 13·0 mg. per cent. and gradually falls to the general level. In the cases of hypomagnesaemia, however, a rapid fall to values as low as 6·0 mg. per cent. often occurred. This was usually associated with the appearance of the earlier clinical signs noted below.

The Effect of Oral Magnesium Supplements on Serum Magnesium and Calcium Levels

At one stage in the first year, two animals in Groups 1, 2, 4 and 5 were each given an oral supplement of 28 g. (1 oz.) magnesium carbonate, equivalent to 8 g. magnesium, daily for one week. They were bled immediately before the supplement was commenced, at 48 hours afterwards, and then at intervals of a few days. Results for the samples taken after 48 hours showed that nearly all serum magnesium levels had been restored to normal in this time. When the supplement was withdrawn, serum magnesium levels fell until within a week they were again well below the normal range. The results are given in Fig. 3. Three of the calves among those given the supplement and three of the controls were showing a hypocalcaemia in addition to hypomagnesaemia when the supplement was commenced. The subsequent rise in serum magnesium levels of the calves given magnesium carbonate was accompanied by a rise of the low serum calcium levels to within the normal range. This effect can be seen in the average levels given in Fig. 3. In a similar experiment, calcium carbonate had no effect.

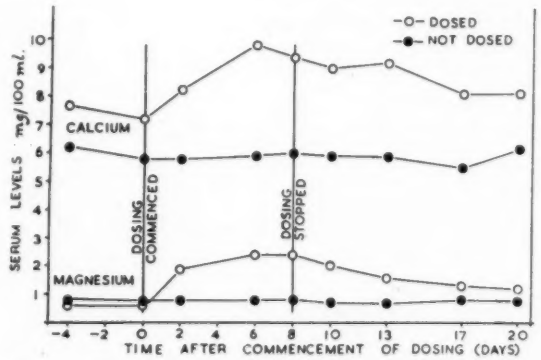

FIG. 3.—The effect of oral magnesium carbonate on the serum calcium and magnesium levels of calves with hypomagnesaemia and concomitant hypocalcaemia.

Blood Pyruvic Acid Levels

The clinical symptoms of hypomagnesaemia resemble in several ways those described for the terminal stages of thiamine deficiency in other animals. This resemblance has also been considered by Blaxter, Rook & MacDonald (1954a). A lack of thiamine interrupts the metabolism of pyruvic acid in the carbohydrate breakdown cycle and consequently animals suffering from thiamine deficiency show increased pyruvic acid levels in the blood and other tissues. Magnesium is also known to be involved at the same stage in the carbohydrate breakdown cycle, and because of this and the similarity in clinical symptoms of the deficiency it was considered worth while to determine whether there was any accumulation of pyruvic acid in the blood of hypomagnesaemic calves. Blood samples were, therefore, taken from normal calves and those with varying degrees of hypomagnesaemia, including animals with clinical symptoms.

The normal pyruvic acid level appeared to be about 0·9 mg. per 100 ml. and this level did not significantly increase until tetany was imminent. Fairly high levels were found, however, for a considerable length of time after convulsions ceased. In one case, a blood sample taken two and three-quarter hours after the cessation of convulsions still showed a level of 3·3 mg. per 100 ml. The increased muscular activity associated with tetany may *per se* cause an increase in the blood pyruvate level, and Blaxter *et al.* (1954b) consider this to be the case. The fact that no significant increase in blood pyruvate levels occurred in these calves until tetany appeared agrees with this view.

Clinical Symptoms

The earliest signs of abnormality were nervousness and a brighter appearance of the eyes. This was followed by increased hypersensitivity, flapping of the ears, champing of the jaws, kicking at the abdomen and swishing of the tail, giving a general agitated appearance as though badly worried by flies, although none was present. Sometimes the animals experienced difficulty in drinking from the buckets and would sway their heads as if trying to determine their exact position. This behaviour probably accounts for reports of blindness which have been received from field cases. In fact there was no evidence of actual blindness, and the difficulty appeared to be due to inco-ordination of the neck and facial muscles. Convulsions were not usually seen until a few days after the appearance of the symptoms described above. Scouring, with dark brown faeces, sometimes preceded the onset of convulsions by one to two days. Convulsions were often precipitated when blood samples were being taken, or at the sound of attendants arriving with the morning feed, in addition to extraneous noise as mentioned above. The convulsions were accompanied by marked exophthalmos, opisthotonus and salivation. In some cases where the symptoms appeared at an early stage in the experiment, the animals recovered spontaneously from the first con-

vulsion, only to succumb to further attacks a few days later. Those which did recover often showed signs of muscular weakness. With increasing age, however, recovery became less frequent, and the older animals died within about 20 to 30 minutes from the onset of the first convulsion.

Post-mortem Findings

Post-mortem examination of fatal cases revealed no distinctive lesions. In some calves there were small haemorrhages in the coronary fat, and on the pericardium, with some larger haemorrhagic areas on the aorta. In one case there were marked haemorrhages along almost the entire length of the alimentary canal. These haemorrhages were not a constant finding and, since their presence or otherwise appeared to depend on the severity of the terminal convulsion, they were probably agonal lesions. In general the severity of the convulsions, and of the *post-mortem* appearance, increased with the age of the calf.

In agreement with the work of Blaxter & Sharman (1955) there was no evidence of calcification of the aorta or of any other tissues from these cases, neither was there any degenerative change of the muscle tissue.

The Magnesium Content of Tissues and Bones

The results of the analyses of tissues and bones are summarised in Tables II and III. The results given are the average values for the animals in each group.

It can be seen that the magnesium contents of the muscles, liver and kidney from hypomagnesaemic calves show no significant difference from those for calves reared on a hay and cake ration and having serum magnesium levels within the normal range. There is, however, a considerable reduction in the magnesium content of the bones from the hypomagnesaemic calves compared with those of normal calves. The magnesium content of the former is about 70 per cent. of that in the normal animals, but the bone magnesium values are not closely correlated with the terminal serum magnesium levels. The period of depletion, depending on the age of the animal, appears to be the predominant factor in determining the level of magnesium in the bones.

Discussion

The results given above show that under the conditions prevailing in these experiments, hypomagnesaemia could be produced in calves by feeding them a ration consisting mainly of whole milk. The magnesium content of the milk varied between 9 and 14 mg. per 100 ml. and there was, therefore, no indication that it was below the usual range. It appears that under certain conditions the magnesium content of milk is insufficient to meet the needs of the growing calf. Male and female calves appeared to be equally susceptible to hypomagnesaemia in these experiments.

TABLE II

THE RESULTS FOR THE FIRST EXPERIMENT

Group	Number of calves examined	Terminal serum levels		Bone ash tibia			Magnesium content of muscle tissue, mg. per 100 g. of fresh weight				Mg in liver per cent. D.M.	Mg in kidney per cent. D.M.	Age at death in weeks		Final body-weight, kg.
		Ca mg. per cent.	Mg mg. per cent.	Ca per cent.	Mg per cent.	Ca : Mg ratio	Quadriceps	Triceps	Psoas	L. dorsi			Individual	Average	
1	4	6·4	0·7	38·1	0·46	83	24·8	22·7	21·8	24·6	0·07	0·08	11, 24, 25‡, 25‡	22	76
2	4	9·1	0·7	38·3	0·45	85	26·1	23·7	25·1	23·6	0·07	0·08	22, 24, 31‡, 31‡	27	130
3	4	7·1	0·8	38·5	0·53	73	24·1	22·7	22·7	21·4	0·14	0·08	13, 14, 20, ‡31	20	85
4	3	8·8	0·7	38·3	0·44	87	25·3	23·2	23·0	24·2	0·09	0·08	25‡, 26‡, 31‡	24	91
5	2	8·0	1·1	38·2	0·59	65	22·7	19·6	18·8	19·4	0·08	0·10	14, 23	18	49
Normal	5	10·2	2·3	38·2	0·62	62	24·2	22·6	23·7	24·2	0·07	0·08	—	14‡	—

‡ Indicates calves were killed at the age shown.

TABLE III

THE RESULTS FOR THE SECOND EXPERIMENT

Group	Number of calves examined	Terminal serum levels		Magnesium content of bone ash								Age at death in weeks		Final body-weight, kg.
				Radius		Tibia		Rib		Thoracic vertebrae				
		Ca mg. per cent.	Mg mg. per cent.	Mg per cent.	Ca : Mg ratio	Mg per cent.	Ca : Mg ratio	Mg per cent.	Ca : Mg ratio	Mg per cent.	Ca : Mg ratio	Individual	Average	
A	4	8·8	1·0	0·46	84	0·52	74	0·46	89	0·38*	100	12, 19, 21‡, 21‡	16	51
B	3	8·5	0·4	0·45	85	0·48	80	0·32	120	0·33†	116	17, 17, 21‡	18	57
C	2	8·7	1·0	0·46	82	0·49	77	0·39	100	0·31†	120	13, 20	17	40
D	3	8·8	0·4	0·63	66	0·57	65	0·45	90	—	—	12, 12, 14	13	44

* Average of two cases only. † One case only. ‡ Indicates the calf was killed at the age shown.

The fact that the needs of the calf for magnesium appear to be greater than those of non-ruminant species has led to considerable speculation as to what factors may be involved in the increased requirement. Sjollema (1935) argued that there was no reason to ascribe tetany in calves to a magnesium deficiency *per se*, and suggested that the disease was due to overfeeding with protein. The results obtained by Wise, Petersen & Gullickson (1940) who fed increasing levels of milk protein, did not support this suggestion. In the present experiments, the addition of a carbohydrate low in magnesium (flaked maize) did not alleviate the fall in serum magnesium as might be expected if a high ratio of protein to energy constituents were involved. If anything it hastened the onset of tetany. This is in agreement with the work of Sharman, Hutcheson & Blaxter (1954) who found that hypomagnesaemia was more common in calves receiving the highest calorie intake. Increasing the level of milk fed to 20 per cent. of the bodyweight daily, and hence the growth rate of the calves in Group 2, did not hasten the onset of tetany but in fact delayed it. In this case the proportion of the various milk constituents to one another and to the magnesium remained constant.

In another discussion of the problem (Wise, Gullickson & Petersen, 1939), it was suggested that a possible lack of one of the vitamin B group may lead to defective utilisation of the dietary magnesium. In addition, the clinical symptoms of pyridoxine deficiency described by Johnson, Pinkos & Burke (1950) resemble in some ways those of hypomagnesaemia. The negative results obtained with the feeding of supplements of the B group of vitamins in these experiments do not suggest that a lack of either of these is involved. Neither do they suggest that an increase in the intake of these vitamins has any sparing effect on the magnesium requirement of the calves. The addition of penicillin to the rations in an attempt to modify the bacterial flora of the alimentary tract likewise failed to have any effect on the course of the hypomagnesaemia.

By the addition of acid extracted hay to the ration for one of the groups (No. 4) it was hoped to determine whether the presence of roughage was essential for the effective utilisation of magnesium. Animals in this group did not show signs of tetany, but the serum magnesium levels fell into the dangerous range. Unfortunately, it was not possible to free the hay of magnesium entirely, so that the additional magnesium intake was probably just sufficient to prevent the occurrence of tetany during the experiment. It seems unlikely that the presence of roughage *per se* in the ration had any effect on the utilisation of magnesium.

Blaxter & Rook (1954), using magnesium balance studies, carried out estimations of the magnesium requirement of calves fed on artificially prepared milk containing various levels of magnesium. They estimated that the calf's dietary requirement of magnesium was 16 to 18 mg. per 100 ml. of milk depending on the rate of growth. On the assumption that the average daily milk intake of a calf is 12 per cent. of its bodyweight, this corresponds to a requirement of 19 to 22 mg. of magnesium per kg. bodyweight, whereas the actual daily amount obtained from whole milk would be about 12 mg. per kg. bodyweight (equivalent to 540 mg. per 100 lb. bodyweight), and these authors concluded therefore that hypomagnesaemia in calves is due to a simple dietary deficiency of magnesium. However, the administration of supplements which supplied 500 mg. of magnesium daily to calves on commercial farms (Blaxter & Sharman, 1955)—that is, approximately doubling the normal intake—did not eliminate hypomagnesaemia, except during the first month of life. The present experiments suggest that mild hypomagnesaemia only would be encountered in the first month of life even of calves receiving no magnesium supplements. This is also borne out by results on serum samples submitted to this laboratory for routine analysis.

It is of interest to compare Blaxter & Sharman's finding with the effect obtained in the present work by the administration of a relatively large amount of magnesium for a shorter period. The calves given 8 g. magnesium daily (as $MgCO_3$) for a week, *i.e.*, a total of 56 g. magnesium, were again showing hypomagnesaemia within seven days of the cessation of the supplement. It is not known for how long this supplement would have to be administered to prevent the subsequent fall.

Another point which is difficult to explain if one accepts the postulate of a simple dietary deficiency is why the condition is not more widespread. There is no evidence that the magnesium content of milk of farms where symptoms are observed is less than on other farms where no tetany occurs. Neither has it been possible so far to establish any consistent difference in the methods of calf husbandry between farms which do, and those which do not, experience the disease. Suggestions that the utilisation and requirements of magnesium in the milk-fed calf are influenced in some way by other factors cannot be ruled out completely on the evidence available. However, the present work does exclude several factors which have been tentatively suggested at various times.

The significance of the fall in serum calcium which occurs in many of the cases is not known. In blood samples, from field cases of calf tetany submitted to this laboratory for examination, the concomitant hypocalcaemia is a constant finding. Blaxter & Sharman (1955) also noted its presence and they suggest that it is due to a metabolic rearrangement and not to a lack of calcium. This view is supported by the results obtained on the administration of magnesium carbonate to the calves described above. It is of interest to recall that Allcroft (1947) found a concomitant hypocalcaemia in 75 per cent. of the blood samples which he examined from cases of hypomagnesaemic tetany in adult bovines.

A depression of the bone magnesium content appears to be characteristic of hypomagnesaemia in calves. Available data (Cunningham, 1936 ; Parr & Allcroft, 1953b) suggest that a depression of the magnesium content of bone ash does not occur in milking cows which have died from lactation tetany. Presumably a part of the magnesium in the bones of calves is present in a form which can be mobilised to counteract the effects of a negative magnesium balance, whereas in older cattle a readily available form is absent. The results for the bones given in Tables II and III indicate that depletion occurs to a greater extent in the ribs and vertebrae than in the long bones, suggesting that magnesium is more labile in cancellous bones. This corresponds with the published information available on the mobility of the calcium which they contain.

The value of bone magnesium levels as an aid to the diagnosis of hypomagnesaemic tetany in calves has already been pointed out by Blaxter (1954), since he calculated that the normal range for the magnesium content of bone ash was 0·55 to 0·75 per cent. The corresponding normal range for the calcium : magnesium ratios was 48 to 70. It is obvious that a certain amount of caution should be exercised in the interpretation of results. As mentioned before, the degree of depletion of magnesium varies from one bone to another in the same animal. This variation probably depends on the length of time during which depletion has occurred. For example, in Table II, of the four calves in Group 1, fed on whole milk only, two died of tetany at 11 and 24 weeks of age, while the other two were slaughtered at 25 weeks of age. The magnesium contents in the ash of the tibia of these animals were 0·52, 0·42, 0·50 and 0·42 per cent. respectively. The first calf to die shows the highest bone magnesium level, even higher than those of the two which were eventually slaughtered without showing clinical symptoms.

A similar effect can be seen in Table III for Group D, also fed on artificial milk. The average age at death was 13 weeks, and the magnesium content of the ash from the tibia was 0·57 per cent. with a calcium : magnesium ratio of 65. The magnesium content for the radius was higher at 0·63 per cent. with a calcium : magnesium ratio of 66. On the other hand the ribs gave 0·45 per cent. magnesium with a calcium : magnesium ratio of 90. The average results for the other groups show a similar trend, with the lowest values being found in the thoracic vertebrae. One case in Group A, however, showed a magnesium content of 0·56 per cent. in the ash of the tibia, 0·56 per cent. in the rib, but 0·45 per cent. in the radius.

At the same time it must be borne in mind that there is no obligate association between low bone magnesium levels and tetany. An animal may have suffered a period of magnesium depletion and hence low bone magnesium values will be found *post mortem* although death may have occurred from another cause.

Summary

1. The experimental production of hypomagnesaemic tetany in calves fed on rations of whole milk and of artificial milk is described.

2. There was no evidence that the magnesium content of the whole milk was below the normal level.

3. It is shown that no preventive effect was obtained by the addition to the milk diet of starch food, roughage, penicillin or of the vitamin B group, which have been suggested as having a possible influence on the utilisation of magnesium.

4. Only mild hypomagnesaemia was observed during the first four weeks of life. Clinical symptoms of tetany became apparent from eight weeks of age onwards.

5. There was a considerable reduction in the magnesium content of the bone ash of nearly all the affected calves. The extent of depletion varied between bones. It appeared to depend partly on the terminal serum magnesium level but more especially on the period of depletion.

6. A supplement of 8 g. magnesium (as $MgCO_3$) daily for a week was sufficient to restore serum magnesium levels to the normal range, but when administration was stopped the magnesium content of the blood fell.

7. The significance of these findings in relation to the causation of hypomagnesaemia in calves is discussed. It is considered that evidence in support of the view that hypomagnesaemia in calves can be directly related to the magnesium content of the milk being consumed is not conclusive, and the influence of other factors cannot be entirely ruled out.

Acknowledgments.—I wish to express my thanks to Dr. Ruth Allcroft and Mr. McGirr for their guidance ; and to Messrs. B. Roberts and L. Rampton for technical assistance.

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THE SUPPLEMENT

An issue of the Supplement to THE VETERINARY RECORD is in preparation, and honorary secretaries of Divisions, or others who have items for publication, are asked to be good enough to forward them not later than Monday, February 4th. This issue will contain the proceedings of the Council of the R.C.V.S. from which items of current interest are quoted this week.

A Case of Acute Copper Oxychloride Poisoning in Cattle

BY
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Coleraine, Northern Ireland
AND
JAMES R. TODD
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THE use of copper compounds in agriculture, notably in the control of parasitic fungi in potatoes and fruit trees, has resulted in sporadic cases of acute copper poisoning, and the toxic effects of various compounds have been studied by different workers from time to time. Kaufmann (1901) quoted 300 grains (about 20 grammes) of copper sulphate as the poisonous dose for the ox. Cunningham (1946), however, reported that yearling heifers and adult cows tolerated repeated doses of 20 to 40 g. of copper sulphate, and single doses of up to 100 g. The lethal dose seemed to lie between 200 and 400 g. The difference between the two figures is striking, though it may be, as with other toxic substances, that the reaction is greatly affected by the clinical state of the animal, and the diet at the time of poisoning.

Sprays such as Bordeaux mixture, based on copper sulphate, are now largely superseded by proprietary preparations; we record below a case in which two cattle died after ingesting a proprietary brand of copper oxychloride.

History

After being driven some four to five miles on a hot day, steers were accidently allowed access to a concentrated suspension of copper oxychloride, prepared for spraying potatoes, and two of the animals drank some of the preparation. The suspension contained 5 lb. copper oxychloride in 2 gallons, and the animals drank a quantity of not more than 1 pint between them, according to the farmer's estimation. The total amount of copper oxychloride ingested was, therefore, about 5 oz. or the equivalent of about 300 g. of copper sulphate.

The steers, of about 11 to 12 cwt. live-weight, ingested the copper oxychloride at 1 p.m., and the owner marked them and watched them carefully for signs of poisoning. One animal showed toxic symptoms 48 hours later and the other about 70 hours later. The condition of the animals rapidly deteriorated, and calcium versenate (6 g. each, intravenously) did not relieve the condition, both animals dying 24 to 36 hours later.

The symptoms included a very severe greenish-blue watery scour (samples showing moisture contents of 91 to 97 per cent), depression, and respiratory difficulty with a very weak, rapid pulse. The onset of the symptoms was quite sudden in each case; at the time of examination of the first animal the second was grazing normally and not scouring. Four hours later the symptoms were as described above.

Samples of faeces and blood were taken from both animals before treatment, and on *post mortem*, liver, kidney and spleen were analysed for copper content.

Post-mortem Findings

These were of an acute enteritis, but without macroscopic evidence of jaundice or other changes in the organs of the liver, kidney and spleen.

Laboratory Findings

The results of the analysis of faeces, of blood and tissue samples from the two steers are set out in Table I below:—

TABLE I
COPPER CONTENTS OF *Ante-* AND *Post-mortem* SAMPLES FROM STEERS POISONED BY COPPER OXYCHLORIDE

Sample	Steer 1	Steer 2	Normal
Ante-mortem			
Blood (μg. per ml.)	1·7	1·6	0·7 to 1·7 (Beck, 1941)
Faeces (μg. per g.D.M.)	10,000	8,000	
Post-mortem			
Liver (μg. per g.D.M.)	64·7	67·4	20 to 200 (Jamieson to Allcroft, 1950)
Kidney („ „)	25·0	11·0	20 (Cunningham, 1931)
Spleen („ „)	14·7	9·9	3 (Cunningham, 1931)

The high concentration of copper in the faeces, amounting to 1.0 per cent. and 0.8 per cent. in the two cases, confirms the ingestion of copper oxychloride in relatively large quantities. The blood levels are at the upper end of the normal range for mature, healthy cattle. Liver values were at the lower end of the normal range. There is no accumulation of copper in the kidney in either case, and though the spleens contained slightly more copper than normal, the amounts of copper involved would be negligible in comparison with the amount ingested. Taken generally, the analytical figures show that the copper was not absorbed to any appreciable extent, which is in keeping with the *post-mortem* findings.

In discussing case histories of copper poisoning in cattle, Diernhofer (1947) distinguishes two types: (a) where the copper is not absorbed, and causes irritation of the digestive tract, and (b) where the

(*Concluded at foot of col. 1 overleaf*)

The Royal College of Veterinary Surgeons

SOME of the principal items of interest arising out of the recent Quarterly Meetings of the Council are set out below.

Irish Universities

Before the commencement of the meeting, the President, Professor L. P. Pugh, welcomed to the Council the two representatives of the Irish Universities, Professor W. J. E. Jessop of the University of Dublin (Trinity College), and Professor P. A. McGeady of the National University of Ireland.

Obituary

Amongst the names especially noticed in the obituary list were those of a past President, Mr. G. P. Male, and a good friend of the Royal College, Sir Lionel Whitby, Regius Professor of Physic in the University of Cambridge. Another name mentioned was that of the late Assistant Registrar, Mr. H. M. Anderson.

The President spoke as follows:—

"Mr. Male was a member of Council for over 30 years and was President in 1931-2 and was well known to you for the meticulous way in which he attended to Council business. I would like to speak of him as I knew him in the field for over 30 years. He was one of the old school—reared on the horse and had spent his whole life among horses. I frequently met him in the clinical field and I had the greatest admiration for the manner in which he brought the art of diagnosis in the horse into his daily work. The authority and confidence he seemed to convey to the owner, the horse, and to me was amazing—especially when he would stand behind a Thoroughbred holding its tail from side to side comparing the muscles of the hind quarters, apparently quite oblivious of the fact that he could be kicked.

"To lunch with him on a horsey occasion was a treat—he was so full of tales of the hunting world over the last 50 years. He will be missed by many.

"Sir Lionel Whitby, as you all know, was Regius Professor of Physic at Cambridge University and Master of Downing College and a haematologist of international repute. Those things you all know, but some of you may not know what a great friend he was to the profession at Cambridge during the inceptive phase of the School. The Institute and subsequent Department of Animal Pathology had been there for 20 odd years and had come to be accepted for the quality of its research work, but it was a different matter when it was agreed that a Department of Veterinary Clinical Studies should be added to constitute a School of Veterinary Medicine, and there was no one more appreciative of the difficulties that we were up against than Sir Lionel Whitby. He realised on the one hand what our feelings were, and he also realised, as a past Vice-Chancellor, the difficulties we were up against from the University side and he fought our battles strongly for us in the Council and before the General Board. I am sure Professor Beveridge will agree with me that we have lost a very good friend both to the Veterinary School and to the profession."

The Registrar spoke of Mr. Anderson on behalf of the staff.

Fellowship by Election

One of the most pleasant parts of the Council meeting was when Fellowship by election was conferred upon Mr. Henry Sumner. In this connexion the President said:—

"The day before yesterday I proposed to the Chairmen's Committee that the Fellowship by election of the Royal College should be conferred upon Mr. Harry Sumner and on that occasion it was passed unanimously.

"The reason why I felt that we should so honour him was for his great service to the profession over the last 45 years. The name Sumner is and has been an honoured one throughout the profession through several generations. He has served the profession in many ways but I would say that one of his greatest efforts has been that he, as a practitioner of the highest standing, has consistently taken a great and

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A Case of Acute Copper Oxychloride Poisoning in Cattle—*Concluded.*

copper is absorbed and nervous and muscular symptoms develop. The *post-mortem* and laboratory findings would put these cases in type (a), which affords an explanation of the failure of calcium versenate treatment. McDonald (1946) showed that intramuscular injection of B.A.L. (dimercaptopropanol) caused greatly increased excretion of copper. Calcium versenate would be expected to act in a similar manner, forming copper versenate which, being non-ionisable, would relieve the metabolic effects of toxic amounts of copper in the blood and tissues. As the copper had not been absorbed, calcium versenate was of no value, but it might be effective in cases of type (b) above, *e.g.* chronic copper poisoning, or in acute cases if it were administered orally within a short time of the ingestion of the copper compound.

Summary

A case is described in which two steers died after ingesting a quantity of copper oxychloride estimated to be about 2½ oz. each. *Post-mortem* and laboratory findings indicated that the copper was not absorbed and that the cause of death was an acute enteritis.

Acknowledgment.—We gratefully acknowledge Mr. R. Pyper's assistance in the laboratory work.

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active interest in furthering the educational side of the profession. This he has done both *via* the University of Liverpool and by his activities as a visitor to the various university veterinary schools. His work for the Lancashire Division of the British Veterinary Association and his interest in veterinary benevolent work are well known.

" Harry Sumner's services came to a climax during his year as President of this College. Members would remember the masterly way in which he had taken all the business which came to him, and the dour fight he had put up in regard to certain situations in Ireland. His success during his term of office decided me that it would be tragic if he was not left to carry on—not exactly the fight—but the business to the point where at last we got a sound basis for the development of veterinary education in Ireland. Mr. Sumner, just as much as the Irish members here, will agree that we have a fair way to go yet before we get a report in this chamber saying that the visitors are entirely satisfied in all branches of veterinary education in Ireland but there is no doubt that a real basis has been laid in Ireland by the work of Mr. Sumner. I put forward the proposition that for his general great services to the profession the Fellowship by election to this College should be awarded to Mr. Sumner."

The proposal was put to the meeting and carried unanimously.

Royal Society of Health

The Registrar was directed to send to Professor Burrow a unanimous resolution of appreciation on his election as Chairman of the Royal Society of Health.

It was noted that the President, Professor Pugh, was President this year of the veterinary section.

Rebuilding of the College

The Council appointed as the architect for this task, should the scheme ultimately take place, Mr. S. H. Moore, the present architect and surveyor to the College. A committee had interviewed various architects, and had seen buildings and plans of buildings designed by them, and unanimously recommended to the Council the appointment of Mr. Moore.

Annual General Meeting, June 5th

The Council gave approval to an entirely new conception relating to the day of the Annual General Meeting (June 5th). In the morning there will be held at the Queen's Chapel of the Savoy a special service. This will be followed by a luncheon at the Savoy Hotel, which in turn will be followed by the Annual General Meeting at the College in the afternoon. It is hoped that the change from a dinner to a lunch, combined with these two ceremonies, may enable more members of the profession to be present.

Fellowship

The Diploma of Fellowship by examination was awarded to four candidates. The names of the candidates, together with the titles of their respective theses, were:—

Mr. Harold Dudley Mills, " The Choledocho-Duodenal Junction with Reference to the Significance of the Sphincter of Oddi."

Dr. Richard Lionel Chandler, " Investigations on Johne's Disease of Cattle and Sheep in New Zealand."

Dr. Howard Henry Holman, " Veterinary Haematology with Special Reference to the Cow and Calf."

Mr. Hugh Percival Harding, " The Histopathology of Johne's Disease in Experimental Animals."

The close of the regulations for the Fellowship under the old By-laws was marked by a great rush of candidates, in all, 27.

There was considerable activity in the field of the Fellowship under the new regulations, and the Council dealt with 10 candidates under the new By-laws.

Hungarian Refugees

It was reported that the Officers of the College met the Officers of the British Veterinary Association regarding a proposition which had been made that a separate fund be established for the relief of Hungarian veterinary surgeons and students. The Council agreed with the joint finding of the Officers that it would not be appropriate to establish a separate fund overlapping with the present Lord Mayor's fund. However, the Council decided to give every help that was possible in establishing Hungarian veterinary surgeons and students within the veterinary schools or in suitable work.

P.D.S.A. Private Bill

A further private Bill presented by the P.D.S.A. to the House of Commons was reported, and it was unanimously resolved that a petition be lodged against the Bill, and that it be opposed in Committee. (The B.V.A. is petitioning against this Bill also, and a note on the subject will be published next week.)

Annual Fees of the Royal College of Veterinary Surgeons in Relation to Schedule E

As it has not been possible to make any progress in this matter through the medium of the Parliamentary and Scientific Committee, although tribute was paid to the work done by the Committee and its Chairman, Sir Hugh Linstead, M.P., of the Pharmaceutical Society, it was decided that the Government should be approached with a view to providing the necessary relief to veterinary surgeons who are assessed under Schedule E in respect of their annual fees to the College.

HORMONE IMPLANTATIONS FOR FATTENING CATTLE

Dr. G. E. Lamming, of the School of Agriculture, Sutton Bonington, recently demonstrated, at the Brackenhurst Farm Institute, a method of hormone implantation in the top of the ear, to speed up the fattening of cattle.

Dr. Lamming said that they had been using between 36 and 120 mg. for cattle implantation. On 14 yard-fed steers, treated with 60 mg. of stilboestrol, there was an average daily gain of 2 lb. Untreated animals gained at an average of 1.22 lb.

Seventeen yard-fed animals treated with 36 mg. showed an average daily gain of 1.09 lb. Untreated ones showed an average daily gain of 0.59 lb.

Twenty-three pasture-fed animals treated with 36 mg. of stilboestrol showed an average daily gain of 2.76 lb., compared with 2.39 lb. average daily gain of untreated cattle.

New Sanatorium for the London Zoological Society

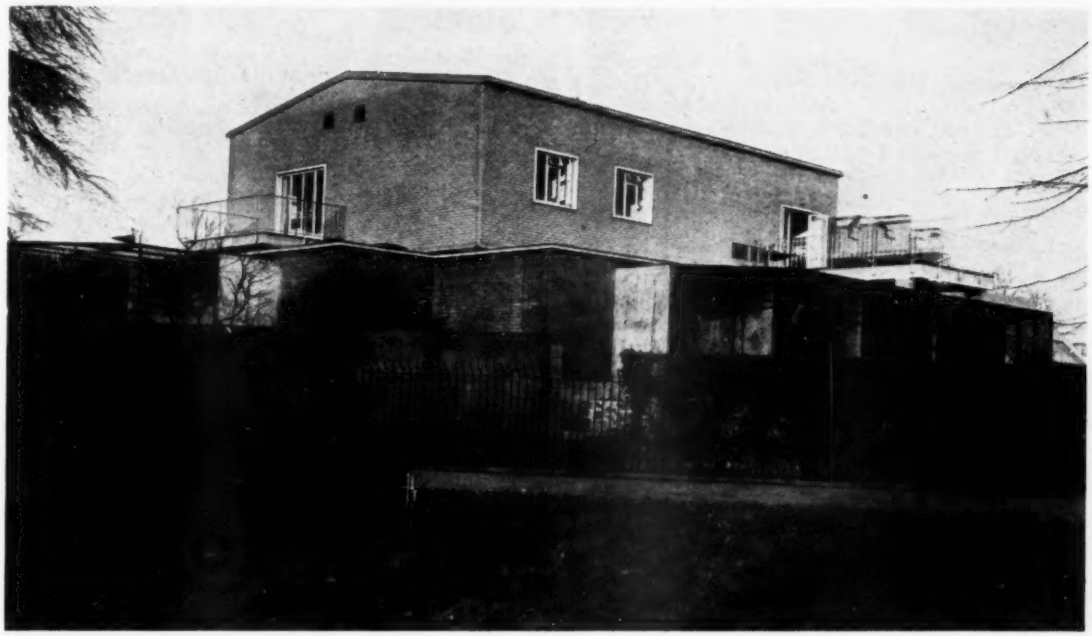
THE Zoological Society of London have established a landmark, not only in their own history but in the history of zoos in general, by opening their new sanatorium. This is the only one of its kind in the world; it has taken nearly two years to build, and has cost the Society £50,000.

On Thursday, January 17th, a Press day was held to display the new sanatorium, and more than 30 journalists and photographers assembled to admire the new building with its pastel walls, huge windows, and general quiet, light-flooded attractiveness. As each journalist arrived he was seized, swathed firmly in a gauze mask, and ushered upstairs to where Mr. Oliver Jones, the Society's Veterinary Officer, was displaying the small-animals' ward to the visitors.

This ward offers every luxury that an ailing animal could desire, including air filters, two warm rooms with the temperature kept at 65° and 75°, infra-red lamps, ultra-violet lamps, and a sun balcony for small basking convalescents. The Press, firing rapid but slightly muffled questions at Mr. Jones through their masks, prowled round the ward taking photographs of the patients, which were mostly monkeys; it was difficult to tell whether the journalists were more fascinated by the monkeys or the monkeys more fascinated by the journalists.

Trooping downstairs, the visitors were shown the spacious operating theatre, the X-ray apparatus, a dazzling kitchen for the preparation of invalid diets, and 21 dens for the larger patients. Mr. Jones's practice at the Zoological Gardens covers everything from a mouse suffering from vitamin deficiency to an elephant with a chill on the kidneys; but he has, regretfully, to refuse the comforts of the sanatorium to the very large animals. Nothing bulkier than lions and full-grown chimpanzees (which are, incidentally, his most difficult patients) can be admitted. The only large patient in the sanatorium to receive the Press was a llama called June, although one journalist was appalled at finding himself suddenly face to face with a capybara, the biggest and surely the most repellent rodent in the world. It had been brought

View at the new hospital, looking from the small-animal treatment ward, out on to the sun balcony.



An exterior view of the new sanatorium.

into the sanatorium to get over a cold in its head. and the journalist complained that it snuffled at him through the bars.

Importance of Preventive Medicine

Even when the new sanatorium gets fully into its stride it is unlikely that all the 21 dens will be filled at once, for the Zoo's sick list, drawn from a collection of 3,000 animals, stays generally at the impressively low figures of 6 to 12 patients. Mr. Jones was emphatic about the importance of preventive medicine, and described it as being far more important than treatment of the sick animals. The therapeutic use of antibiotics and modern chemotherapeutics have assisted in bringing about an over-all fall in the death rate.

Mr. Jones was nonchalant about the problem of injecting the dangerous animals, including the big cats. "They're quite tame," he said. "I just call them up to the bars, stroke them, and give the injection."

Many Zoo ailments are intestinal, due chiefly to the misplaced and obstinate kindness of the public in giving the animals food. When a newly caught animal arrives at the Zoo it displays a sure instinct over its food. It is offered a choice of 25 or 30 different varieties, and will unhesitatingly reject what is bad for it and eat only what is good. But evil communications also corrupt good sense; the example of its neighbours in begging for scraps is too much, and soon it, too, begins to accept the proffered dainties. Monkeys, in particular, are given mouldy bread, onions, unripe or overripe windfalls, chop bones, and even bottle tops. Sometimes food is infected; for instance, a child with a streptococcal throat infection will eat half a sandwich, get tired of it, and give the other half, ripe with streptococci, to an unfortunate monkey. Not long ago a monkey cut its own tongue half out with an open penknife someone had handed to it.

Bank Holidays, bringing as they do enormous crowds and consequently an enormous number of upset digestions, are the bane of Mr. Jones's life, and every animal in the Zoo is given an aperient on the day before the holiday to mitigate the ill effects.

The Sanatorium Staff

The new sanatorium is likely to prove useful to other Zoos, which have not built their own. The staff consists of the Veterinary Officer, a Veterinary Pathologist, five sanatorium assistants, and three technicians, and there is a large laboratory attached, for every animal which dies is subjected to *post-moriem* examination as a routine. Small, delicate animals from hot climates can now spend part of their six weeks' quarantine in the sanatorium's warm rooms, gradually hardening off against the all-the-year-round rigours of the English climate.

At the end of the Press reception (with Mr. Jones looking a trifle exhausted after two hours' intensive questioning) the visitors were given tea, and Sir Solly Zuckerman told them a story about a rhinoceros which had unaccountably gone lame and was being treated with massage. And incidentally, the Press was told about the old sanatorium, now too small and old-fashioned for the Society's needs. After more than 50 years of honourable service, it has lapsed into frivolity and become a television studio.

News and Comment

UNIVERSITY NEWS

Liverpool

B.V.Sc. Second Examination, January 1957, Part I: Veterinary Anatomy (including Histology and Embryology)

Aspinall, Kathleen M.; Bird, Christine M.; Butterfield, W. E.; Callender, D. E.; Capron, T. K.; Chesworth, A. T.; Fitton, J.; Hamilton, J. G.; Holt, H. J.; James, W.; Jenkins, J. T.; McMurtry, M. J.; Michael, H. P.; Miller, J. K.; Nyavor, A.; Pickup, Pamela; Quartey-Cofie, J. E.; Schofield, J. B.; Wild, G. S. P.; Worthington, P. T.; Zanetti, P. J.

Part II: Physiology (including Biochemistry)

Aspinall, Kathleen M.; Bird, Christine M.; Butterfield, W. E.; Callender, D. E.; Fitton, J.; James, W.; Jenkins, J. T.; McMurtry, M. J.; Michael, H. P.; Nyavor, A.; Schofield, J. B.; Wild, G. S. P.; Worthington, P. T.

THE INSTITUTION OF PROFESSIONAL CIVIL SERVANTS

The Institution's Annual Delegate Conference takes place this year at the Winter Garden, Eastbourne, on May 20th, 21st, and 22nd. There will be some 300 delegates present, representing all the professional, scientific, and technical classes in the Civil Service.

NORTH WALES DIVISION ANNUAL DANCE

The annual dance of the North Wales Division was held on December 14th last at St. George's Hotel, Llandudno, when 93 members and guests gathered for a most enjoyable evening. The President of the B.V.A. and Mrs. Callender were the guests of honour, together with Mr. H. Morris Roberts, High Sheriff of Caernarvonshire, and Mrs. Roberts.

In the unavoidable absence of the Division's President, Mr. D. T. Roberts, the senior vice-President, Mr. George Wyse of Daventry, replied to the toast of the Division, proposed by the High Sheriff. The President of the B.V.A. replied on behalf of the Association. Mr. J. Richards proposed the toast of the guests and ladies, to which Mrs. A. Sutherland of Bangor responded.

In this picture are seen, left to right, Mr. and Mrs. Wyse, Mr. and Mrs. Callender, and Mr. and Mrs. Morris Roberts.

PROFESSOR W. M. MITCHELL

As we go to press it is learned that the University Court of Edinburgh have received intimation of the resignation of Professor W. M. Mitchell, Director of Veterinary Education at the Royal (Dick) School of Veterinary Studies. Professor Mitchell's retirement from office will take effect at the end of September this year.

He became Principal of the college in 1946, having been Professor of Surgery since 1919. Between 1910 and 1919 he was assistant to Dr. O. Charnock Bradley. He has had a long association with the B.V.A., being President in 1946-7. He became a member of the Council of the Royal College of Veterinary Surgeons in 1945, and was elected an Honorary Fellow in 1953.

A review of Professor Mitchell's career and services to the profession will be published when his retirement becomes effective.

COLONIAL OFFICE APPOINTMENTS

Promotions

Mr. A. D. Campbell, Veterinary Officer, Eastern Region, Nigeria, has been appointed Principal Veterinary Officer, Eastern Region, Nigeria; Mr. D. C. Hague, Veterinary Officer, Kenya, has been appointed Principal Veterinary Officer, Kenya; and Mr. R. L. Wooldridge, D.F.C., Veterinary Officer, Kenya, becomes Principal Veterinary Officer, Kenya.

New appointments

Mr. D. G. Fanshawe, B.SC., Veterinary Investigation Officer, Northern Nigeria; Mr. J. B. Peters, Veterinary Officer, Uganda; Mr. V. W. Smith, B.SC., Veterinary Research Officer, Federation of Nigeria.

THE REGISTER OF VETERINARY SURGEONS

Under the provisions of Sec. 13 (1) of the Veterinary surgeons Act, 1881, Mr. Spencer Brian Friedman, B.V.SC. (Pretoria), c/o South Africa House, Trafalgar Square, W. C. 2, was admitted to the Commonwealth and Colonial List on January 18th, 1957.

PERSONAL

Births

BOUVET.—On January 11th, 1957, to Honor, wife of R. M. Bouvet, M.R C.V.S., 63, Lichfield Road, Sutton Coldfield, Warwicks, a son, Nicolas James, brother to John Peter and Joanna.

LUCKIN.—On January 15th, 1957, to Sheila, wife of K. G. Luckin, M.R.C.V.S., 205, High Street, Waltham Cross, a son, Stephen Vincent.

KIMBERLEY.—On January 19th, 1957, at St. Asaph Maternity Hospital, to Vicky (*née* Hughes), wife of H. M. Kimberley, M.A., PH.D., a son, David Oliver.

RAMACHANDRAN.—On November 13th, 1956, at the Christian Mission Hospital, Ranipet, to Ramani, wife of P. K. Ramachandran, B.SC., B.V.SC., M.R.C.V.S., a son, Krishna Iyer.

SIM.—On December 26th, 1956, to Dorothy, wife of Lewis Sim, M.R.C.V.S., of Meadfort Cumnock, Ayrshire, a third son.

WARDE.—On January 17th, 1957, at Battle Hospital, Reading, to Phyllis, wife of John D. Warde, M.R.C.V.S., Newbury, a daughter.

Marriage

PODD—BARNES.—On January 5th, 1957, at St. John's Church, Roundhay, Leeds, Geoffrey Harold Podd, M.R.C.V.S., of Kessingland, Suffolk, to Pamela Cole Barnes, B.SC., M.R.C.V.S., of Leeds, Yorkshire.

COMING EVENTS

January

30th (Wed.). General Meeting of the Lancashire Veterinary Association at the Midland Hotel, Manchester, 2.30 p.m.

Ordinary General Meeting of the Sussex Veterinary Society at the Old Ship Hotel, Brighton, 2.30 p.m.

February

1st (Fri.). Annual General Meeting of the Mid-West Veterinary Association at the Berkeley Café, 2.30 p.m.

Annual General Meeting of the Northern Ireland Veterinary Association at the Union Hotel, Donegall Square, Belfast, 6.30 p.m.

6th (Wed.). General Meeting of the Ayrshire Veterinary Association in the Hotel Dalblair, Ayr, 2.30 p.m.

7th (Thurs.). General Meeting of the Central Veterinary Society at the Royal Veterinary College, Camden Town, London, N.W.1, 6 p.m.

13th (Wed.). General Meeting of the Society of Practising Veterinary Surgeons at the Royal Veterinary College, Camden Town, N.W.1, 2.30 p.m.

15th (Fri.). Annual General Meeting of the Royal Counties Veterinary Association at the Caversham Bridge Hotel, Reading, 2.15 p.m.

19th (Tues.). 81st Annual General Meeting of the Eastern Counties Veterinary Society at the Bell Hotel, Norwich, 11.15 a.m.

21st (Thurs.). Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 8.30 p.m.

22nd (Fri.). Annual Ball of the Liverpool University Veterinary Society in the Students' Union, 8 p.m.

March

2nd (Sat.). Meeting of the Scottish Regional Group of the A.V.T. & R.Ws. at the University of Glasgow Veterinary Hospital, 10.15 a.m.

30th (Sat.). Annual Convention of the Ceylon Veterinary Association in the Queen's Hotel, Kandy, Ceylon.

FOOT-AND-MOUTH DISEASE

The area described in the schedule below shall cease to be an Infected Area for the purpose of preventing the spread of foot-and-mouth disease on January 30th, 1957:—

IN THE COUNTY OF OXFORD

In the Petty Sessional Division of Bampton West: The parishes of Taynton, Westwell and Holwell and so much of the parish of Burford and Upton and Signet as lies to the west of the main road (A.361) running from Fulbrook *via* Burford to Filkins excluding the said road.

IN THE COUNTY OF GLOUCESTER

In the Petty Sessional Division of Stow-on-the-Wold: The parishes of Bourton-on-the-Water, Clapton, Little Rissington and Great Rissington.

In the Petty Sessional Division of Northleach: The parishes of Barrington, Windrush, Sherborne, Aldsworth, Farmington, Northleach with Eastington, Hampnett, Turkdean and Aston Blank and so much of the parish of Coln St. Dennis as lies to the north and east of the road running from Ablington *via* Winson and Coln St. Dennis to Chedworth including the said road.

In the Petty Sessional Division of Fairford: The parishes of Eastleach and Coln St. Aldwyn and so much of the parish of Bibury as lies to the north and east of the road running from Coln St. Aldwyn *via* Bibury and Ablington to Winson including the said road.

The area described in the schedule below shall cease to be an Infected Area for the purpose of preventing the spread of foot-and-mouth disease on February 1st, 1957:—

IN THE COUNTY OF ESSEX

The Borough of Maldon.

In the Petty Sessional Division of Dengie and Maldon: The parishes of Woodham Mortimer, Hazeleigh, Purleigh, Cold Norton, Stow Maries, North Fambridge, Mundon, Latchingdon, Althorne, Mayland, Steeple, St. Lawrence, Asheldham, Southminster, Burnham, Dengie, Tillingham and Bradwell-on-Sea.

In the Petty Sessional Division of Chelmsford: The parishes of Woodham Ferrers and Rettendon.

In the Petty Sessional Division of Rochford: The parishes of Canewdon Paglesham, Stambridge, Rochford, Ashingdon, Hawkwell, Hockley and Rawreth and so much of the parish of Rayleigh as lies to the north of the main road (A127) running from Romford to Southend-on-Sea excluding the said road.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak

Anthrax

Durham. 32, Copley Bent, Butterknowle, Bishop Auckland (Jan. 16).

Hants. Sutton Manor Farm, Bishops Sutton, Alresford (Jan. 14).

Hereford. Brook Farm, Little Marele, Ledbury (Jan. 14).

Lanarks. Rutherend Farm, Chapleton, Strathaven (Jan. 17).

Yorks. Stacey Bank Farm, Loxley, Sheffield (Jan 14).

Fowl Pest

Cambs. Bury Farm, Meldreth (Jan. 15).

Essex. Riversleigh, Nipsells Chase, Mayland, Chelmsford; Restbrook, Rectory Lane, Latchingdon (Jan. 14).

Lancs. Lane End Farm New Road, Bretherton; Field end of Tayler's Lane, Pilling (Jan. 14); Halls Cross, Freckleton (Jan. 16); Farrers Farm, Grange Lane, Hutton (Jan. 17).

Norfolk. The Car Dump, Scarning, Dereham; Common Farm, Kenninghall, Norwich (Jan. 16).

Northumberland. 3-9, St. Lawrence Road, Newcastle upon Tyne, 6 (Jan. 14).

Swine Fever

Dorset. Whitecliff Farm, Swanage (Jan. 17).
Hants. Chapel Lane, East End, Lymington (Jan. 17).
Staffs. George and Dragon Inn, Main Street, Alrewas, Burton-on-Trent (Jan. 17).
Surrey. Piggeries, 5th Avenue, Ewell (Jan. 17).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

The Sensitivity of *Bacterium coli* to Chemotherapeutic Agents

Sir,—The importance of the conclusions drawn by Dr. Williams Smith and Mr. Crabb in their paper on *Bacterium coli* resistance to the tetracyclines must be obvious to all.

In a matter of such far-reaching importance to the future health of stock and possibly man, the farming community and medical profession should be made aware of the facts at the earliest opportunity.

I suggest such a statement be made by the Royal College or the B.V.A., for as it may well give rise to dispute amongst interested parties, authority and impartiality are essential.

Yours faithfully,
A. B. BROOK.

39, Southgate,
Chichester,
Sussex.

January 18th, 1957.

[Copies of THE VETERINARY RECORD are sent every week to the medical and agricultural press and to a number of leading newspapers.—*Editor.*]

Acid Firing

Sir,—I was glad to read in THE VETERINARY RECORD of January 12th of yet another veterinary surgeon who is an advocate of acid firing, in the person of Mr. E. F. Beckett.

The method I employ may seem unnecessarily laborious and complicated to one who has not used it, but in fact it is quite simple and enables even an inexperienced operator to fire a leg in a few minutes; the average time taken to apply the sleek and fire a leg is about 10 minutes.

The use of the perforated plaster has the following advantages:—

(a) It ensures that the acid is applied only to the exact spots of skin showing through the holes punched in the sleek and avoids any danger of acid running down the leg.

(b) When firing without the use of plaster it is extremely difficult, when applying the acid for the second and third time, to see the spots already fired and ensure that the acid is applied accurately to them. If two or more spots coalesce it may result in sloughing of the skin.

(c) It gives the operator much greater confidence and enables him to fire more severely as he knows that the spots fired are so arranged that each is separated from its fellow by sufficient live skin to prevent sloughing.

(d) It results in a symmetrical pattern impossible to attain when firing without its use.

By using felt instead of cork, one can fire much more quickly and ensure a more uniform application of acid.

Like Mr. Beckett, I have used acid firing with considerable success in the treatment of curbs, and another veterinary surgeon has written to me saying that he regards it as a specific for bog spavin and thoroughpin; in ostitic conditions such as ringbone and bone spavin I do not consider that it is indicated.

One naturally avoids the use of a twitch whenever possible, more especially in T.B. horses; when employed by me it is applied gently with the object of distracting the animal's attention, and in many cases it is unnecessary.

Mr. J. F. Rankin reports in THE VETERINARY RECORD of January 19th that he has had difficulty in getting the sleek to stick to the leg.

This may be caused by not preparing the leg properly, *i.e.* hair not completely removed by the depilatory paste, or applying the sleek when the leg is damp. Soiling of the sticky side of the sleek may also detract from its adhesiveness; it should be freshly prepared for each case and rolled back on to some kind of large cylinder.

If the hair is completely removed and the leg dried the sleek will stick firmly.

Yours faithfully,
E. S. W. PEATT.

The Lilacs,
Summer Hill,
Ashford, Kent.

January 21st, 1957.

Adrian Jones, M.V.O.

Sir,—Reading about Adrian Jones provokes in me the naughty query: "What has happened to our money?" For in a dimming past many of us were persuaded to subscribe to a fund which was to purchase, cast, and erect a major work of this great sculptor-artist in the grounds of the Royal Veterinary College, Camden Town.

If trustees of this fund still exist, will they please, after rubbing their eyes and taking a cold plunge, consider the possibility of an appeal to the profession with a view to the erection of this master-work of Adrian Jones as part of the adornments of a rebuilt R.C.V.S. in Red Lion Square? Having done this they should have plenty on their hands to inhibit the resumption of their already prolonged hibernation-cum-aestivation in this matter.

Yours faithfully,
R. C. G. HANCOCK.

Meadham Cottage,
Hannington,
Basingstoke, Hants.

January 12th, 1957.

"Dry Coat" in Horses

Sir,—I read the article on "'Dry Coat' in Horses" published in THE VETERINARY RECORD of January 5th with great interest. As a paper written by me and published in the *R.A.V.C. Journal* in 1938 was included among the references, I may perhaps be permitted to make some comments.

The present paper appears to be based mainly on experience of Malayan conditions and to differ in some respects from the appearances noticed in India, where the temperatures are higher, together with at least equal humidity. For example, it would not be correct to say that race-horses in India are especially prone to dry coat during the period of acclimatisation. At that time, they are normally on a moderate ration in their comparatively cool summer quarters, and are doing no fast work. Again, race-horses do not, or did not in my time, receive a high protein ration. The emphasis, indeed, was (and in the nature of things where energy was the first requirement had to be) on a high carbohydrate ration, including frequent reinforcement with glucose. This, with fast work in training and racing, produced considerable internal heat which, combined with the high external temperature, resulted in vulnerable animals in an inability to sweat, and thereby to obtain relief.

In undertaking the considerable amount of work incurred the authors were obviously seeking the cause of this condition, but they appear to agree they have not produced sufficient evidence to prove their case. They have, however, indicated that the increased production of adrenalin may, perhaps, be a result of dry coat rather than the cause. In this they would be in agreement with MacGregor, whose work was mainly directed to demonstrate similar physiological and pathological abnormalities.

On the other hand they have provided evidence indicative of the real cause in their first paragraph, under the heading "Introduction." This is that animals, reared for centuries in the tropical zone, have provided their descendants with an inherited ability to deal with conditions of high temperature and humidity which cannot be supported by a proportion of Thoroughbred horses imported from a temperate zone and subjected, in their new semi-tropical environment, to the strain of continued training and racing. The result is a breakdown in their system of heat control.

That all such imported Thoroughbred horses do not go dry is, with reasonable certainty, due to their descent in the male line from three horses of Eastern blood, the Darley Arabian, the Byerley Turk, and the Godolphin Arabian, all of which derived from tropical desert countries, and in the female line, for the most part, from large numbers of mares of the same origin imported to England from the 16th century onwards. To this day their descendants still retain a considerable ability to stand great heat and humidity.

When this thesis is supported by the fact that a precisely similar condition occurs in European cattle when subjected to tropical heat (though in far greater proportion and much greater severity than in horses), there can be little remaining doubt as to the cause. In these cases the symptoms are a rough dry coat, frequently continuous temperature, and respiration up to 120 times per minute with protrusion of the tongue, etc. The most exaggerated symptoms are seen when high yielding cattle, on a big concentrate ration, are affected, though when these are dry and on a "roughage diet," these severe symptoms are absent or considerably ameliorated.

I had this problem among cattle to deal with in the Indian military dairy farms and drew attention to the fact that up-grading of the indigenous breed by the use of Friesian bulls was resulting in a position where, with each upward grade, the over-all production of milk of that grade showed a regular decline and the cattle themselves were much more susceptible to tropical diseases than pure indigenous stock. It was pointed out that this situation could only be due to a gradual breakdown of the power to control body heat, and to a disastrous dilution of the resistance to tropical diseases. In this connexion the writer has never seen indigenous cattle exhibiting these symptoms.

At the time when India was engaged in breaking down a hereditary resistance in this way, the Santa Gertrudis division of the enormous King Ranch in Texas were using Brahma bulls, the product of India, to build up heat resistance in their Shorthorn cattle. They have, since then, succeeded in developing a true breeding type of cattle, three-eighths Brahma and five-eighths Shorthorn, which is resistant to the severely hot climate of Southern Texas around the Gulf of Mexico and which produces high-class beef.

Should any of your readers wish to pursue this line of thought further, an essay I wrote in 1943, but not regarded as suitable material for the *R.A.V.C. Journal* in war time, was resuscitated and was recently published in the September and October numbers of the *Irish Veterinary Journal*, 1956.

Yours faithfully,

C. M. STEWART.

Dowdstown House,
Maynooth,
Co. Kildare,
Ireland.

January 17th, 1957.

Petrol Rationing

Sir,—Most of us have been granted about 50 per cent. of our individual petrol requirements. A recent opinion from a liaison officer suggests that we may not get any more for the present period. As our clients—the farmers—seem to have as much as they want, one feels our case may not be so hopeless as this opinion would suggest.

The President of the R.C.V.S. has asked us to use our extra ration with intelligent economy. Unlike previous correspondents (*Vet. Rec.*, December 29th, 1956), one feels the request is reasonable, as, normally, the economic use of petrol is hardly ever considered. How often do we spend an afternoon seeing chronic cases in the same district we have travelled in the morning? Have we not asked an

assistant to go—about 10.30 p.m.—to someone who has just found a small patch of wet eczema on a cat? We pride ourselves on the service we give—any animal will be seen at any time. Surely an appeal for economy should be made to the people *for* whom, not *by* whom, the petrol is used?

One wonders what steps we are taking to carry out the request of the President. How many of us have circularised our clients, asking for all non-urgent calls to be in before 9 a.m.? If, Sir, evidence can be produced of at least 10 practices in which this has been done, I would be delighted to hear it—so delighted that I would be prepared to subscribe £3 3s. (all I can afford) to the V.V.B.F. if it is a fact.

At very little expense the Divisions of the B.V.A. could follow the example of A.I. centres and insert an appeal in the local press for all cases to be in by 9 a.m.*

May I make a special request to the Parliamentary and Public Relations Committee of the B.V.A. that they might consider what steps they can take to let the general public know our position? At present it is assumed—owing to statements in the national press—that we can have all the petrol we need.

Yours faithfully,

J. E. JOHNSON.

2, Carlton Street,
Welford Road,
Leicester.

January 12th, 1957.

* [Appeals have already been sent to the press all over the country.—*Editor.*]

Sir,—I had hoped and expected to read, by now, some reference in THE RECORD to the rationing and increased price of petrol, as affecting veterinary surgeons.

All the veterinary surgeons to whom I have spoken have received only about one half of the supplementary ration for which they applied. May we assume that as we are in the "priority" class we shall receive a further supply, on application, when our present stocks are becoming exhausted?

All practitioners will, of course, have to increase their mileage charges to their private clients; but my concern is the Ministry's mileage allowance to the L.V.I. I hope that the B.V.A. have taken this matter up with the Ministry of Agriculture, as a matter of urgency, and that a decision will be announced in time to incorporate the new rates when sending in our January accounts. I would suggest that the allowance should be increased to one shilling a mile, retrospective to January 1st, 1957.

Yours faithfully,

J. V. S. RUTTER.

Gwynne House,
Hereford.

January 12th, 1957.

[References to petrol rationing were made in our issues of December 1st, 8th, and 29th, January 5th and 19th.—*Editor.*]

Methods of Injection

Sir,—During the past few years, it has come to my notice that many veterinary surgeons, when carrying out injections on dogs, invariably give these intramuscularly. The result of adopting this method undoubtedly causes the patient considerable pain and discomfort and, incidentally, causes dissatisfaction on the part of the owner. I was taught, and have noted in several books of reference, that the subcutaneous method is the right one. What, then, is the reason for this method being now out of fashion? Am I right in assuming that the effectiveness of the vaccine or drug employed is in no way altered, whether the method be the subcutaneous or intramuscular?

Yours faithfully,

DENYS DANBY.

55, Elizabeth Street,
London, S.W.1.

January 20th, 1957.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st December, 1956		15	—	7	72	—	23
Corresponding period in	1955	17	1	2	137	—	31
	1954	25	—	—	38	—	68
	1953	20	—	1	52	—	60
1st January to 31st December, 1956		1,245	11	162	956	—	741
Corresponding period in	1955	764	8	9	906	—	1,403
	1954	350	4	12	795	—	1,455
	1953	609	—	40	978	—	2,713